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A Hospital-based Study of Pyogenic Meningitis in Children in Small Urban and Rural Areas of Edo State

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Summary

Akpede GO, Dawodu SO, Umoffia ME, Iyasere GEA, Ukpomwan EMA. A Hospital-based Study of Pyogenic Meningitis in Children in Small Urban and Rural Areas of Edo State. *Nigerian Journal of Paediatrics* **2000; 27: 54.** The incidence, aetiological agents and factors influencing the outcome of pyogenic meningitis in hospitalised children in Edo Central Senatorial Zone are described. The incidence was 48.3 cases per thousand admissions of one month to 15 years old children. This was not only relatively high, but the usual decline in incidence with age was not observed. The other characteristic features were a high incidence of subacute and late presentation and gram negative bacillary meningitis. The case fatality rate was 113.4 per thousand cases. Among the survivors, 174.4 per thousand cases had neurological sequelae. The factors associated with an adverse outcome were the same as those reported from other areas. Further education of all categories of health care workers but, especially of physicians and nurses in general and private practice is required on the recognition and management of pyogenic meningitis, including the need for early referral of suspected cases. This is necessary to improve outcome.

Introduction

FOR various reasons, data on pyogenic meningitis (PM) in the tropics consist largely of reports from hospitals in large urban areas where academic medical centres/teaching hospitals are usually sited. There is thus, likely to be a bias in the reporting of various aspects of PM, which is also of major health concern in some urban and rural areas where a large proportion of the population in most developing countries reside. As far as we are aware, there is a paucity of information on the nature of PM in these areas in tropical Africa. In view of this, and in order to provide some relevant data, this communication reports a study whose aims were to define the incidence, patterns of presentation, aetiological agents, problems in diagnosis, and factors influencing outcome of PM as it affects children living in small urban towns and rural areas.

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Patients and Methods

Site of study

Otibhor Okhae Teaching Hospital (OOTH) which was opened in 1993, is a 242-bed hospital in Irrua, a small town in the central senatorial zone of Edo State. Irrua (pop. 65,000, 1991 census) is about 100 km east-north-east of Benin City (pop. 1,300,000), the capital of Edo State. The hospital is open to all patients, whether self- or physician-referred. The catchment area includes the central and northern senatorial zones of Edo State and parts of the neighbouring states but most of the patients are from the central senatorial zone which is within the northern part of the rainforest belt. There are five Local Government Areas (LGAs) in Edo central sena-

torial zone. However, most (83 percent) of the patients in this study were from two contiguous LGAs, Esan Central LGA with headquarters in Irrua and Esan West LGA with headquarters in Ekpoma. Most of the towns and villages in the two LGAs, including the headquarters, lack pipe-borne water and are dependent mainly on rain water and unprocessed surface water. However, the two LGAs have electricity supply. Malaria transmission occurs throughout the year, but with intensification during the rainy season. A majority of the residents are farmers and traders. The towns and villages in the two LGAs are rural.[1] However, the headquarters, especially Ekpoma which is host to Ambrose Alli University, are urbanising rapidly with the presence of tertiary institutions.

The study, other than the aspect that deals with incidence, involved patients aged one month to 15 years with a diagnosis of PM who were admitted from 17/5/93 to 30/11/99. Patients who had tuberculous meningitis were excluded as were patients whose clinical diagnosis of PM could not be confirmed. The aspect of the study on the incidence of PM was based only on the period 1/1/95 to 30/11/99, due to the fact that it was only from 1/1/95 that concerted effort was made to document in the admission register, discharge diagnosis from which the incidence rate was calculated.

The information, obtained on a prospective basis, included clinical presentation, laboratory data, treatment and outcome. Cerebrospinal fluid (CSF) samples were processed using standard techniques. The diagnosis of PM was based on CSF examination (turbidity, pleocytosis of >10/mm^3 with neutropphil predominance, and chemical changes of CSF/blood glucose ratio <50 percent and protein >80 mg/dl) and typical clinical course. Neutrophil predominance in the CSF was defined as neutrophil count >50 percent of the white blood cell count.[2] Patients with bacterial pathogens identified on Gram stain or culture of the CSF or from culture of blood, were classified as having bacteriologically confirmed meningitis (BCM). Those without identified pathogens were regarded as having bacteriologically unconfirmed meningitis (BUM).

Other investigations included complete blood counts, blood cultures and thick blood smears for malaria parasites. Malaria parasitaemia was graded on a semi-quantitative scale of + to ++++ as described by Bruce-Chwatt.[3] All the tests could not be done in all the patients because the materials were not always available or the parents could not bear the extra cost.

The antibiotic regimen iincludeed chloramphenicol only (n=41), chloramhenicol plus ampicillin or

Table I

Incidence of Pyogenic Meningitis at Various Age Groups (1/1/95 to 30/11/99)

Age	*No Admitted*	*Incidence (%) of Meningitis*		
		No (%) of All Cases	*No (%) of Confirmed Cases*$^+$	*No (%) of Unconfired Cases*$^{++}$
1-6 mon	195	11 (5.6)	4 (2.0)	7 (3.6)
7-12 mon	254	5 (2.0)	0	5 (2.0)
13-24 mon	320	9 (2.8)	3 (0.9)	6 (1.9)
25-60 mon	387	13 (3.4)	6 (1.6)	7 (1.8)
61 mon-10 yrs	223	22 (9.9)	7 (3.2)	15 (6.7)
>10 yrs	296	23 (7.8)	9 (3.1)	14 (4.7)
Total	1675	83 (5.0)	29 (1.8)	54 (3.2)
P-values*	-	<0.0001	0.043	0.005

Note: The total number of patients is less than the 1719 stated in the Text because the ages of some patients were not recorded.
$^+$Positive Gram stain/culture of CSF or positive blood culture
$^{++}$Negative Gram stain/culture of CSF or negative blood culture where this was done
*X^2 test with 5df.

penicillin (n=32), ampicillin or penicillin (n=13), the newer cephalosporins (n=6) and cloxacillin plus gentamicin (n=44) given in standard doses.[4] Six patients died before they could be commenced on adequate doses of antibiotics; two patients were receiving inappropriate antibiotics while four had not been commenced on any antibiotics at all. Other treatment modes included control of convulsions, medical measures to reduce intracranial pressure/cerebral oedema, and appropriate fluid restrictions. The patients were reviewed frequently initially and at least, once daily after stabilisation, until discharge. Follow-up was in the Neurology Clinic which was held once weekly.

The patients were retrospectively scored for the severity of illness at presentation using the method of Akpede et al.[5] By this method,[5] a score of one is given for the presence of each of 10 clinical features: age <2 yrs, illness >7 days, pre-admission antibiotic therapy, focal nerve deficits, abnormal posturing, abnormal muscle tone, lack of typical meningeal signs, shock, unrousable coma and seizures. A patient with an aggregate score of three or more, was regarded as being severely ill.[5]

Statistical analysis was carried out, using Epi Info Version 5;[6] chi squared test with Yates' correction for continuity or Fisher's Exact Test, was used for tests of significance, as appropriate. Two-tailed p-values less than 0.05 were taken as significant.

Results

One hundred and two patients fulfilled the criteria for the diagnosis of PM. Forty-five patients (44.1 percent) had BCM while 57 (55.9 percent) had BUM. Twenty-nine patients were from Esan Central LGA, 59 from Esan West LGA and nine from other LGAs in Edo central senatorial zone while four patients were from other LGAs in Edo State and one patient was from a neighbouring state. Among the 29 patients from Esan Central LGA, 15 were from Irrua while 14 were from other towns and villages. Among the 59 patients from Esan West LGA, 44 were from Ekpoma while 15 were from other towns and villages.

Incidence of bacterial meningitis

Eighty three (48.3 per 1000 admissions) of 1719 infants and children who were admitted from 1/1/95 to 30/11/99 had PM (16.87 per 1000 with BCM and 31.41 per 1000 with BUM). There was no significant seasonal variation in incidence. The overall incidence of PM and the incidence of BCM and BUM varied significantly with age as shown in Table I. The overall incidence PM was lowest in the second half of infancy; it thereafter, increased with age and remained high in late school age.

Table II

Pre-admission Consultations in 102 Patients with Meningitis

Personnel/Places Consulted	*No of Cases*	*Percent of Total*
Hospital*/physicians in private practice**	57	55.8
Maternity homes, health centres, nurses in Private practice+	7	6.9
Physician and nurse, consulted separately	2	2.0
Patent medicine/chemist shops++ only	17	16.7
Herbalists only	9	8.8
None/self-medication	10	9.8
Total	102	100

* Public or private general hospitals manned by "general practice Physicians" who usually have no postgraduate diploma

** Physicians with unregistered home-practices

\+ Usually manned by nurses/midwives

++ Usually manned by persons without any formal education in health/medical care but nurses and other on-physician personnel have also become increasingly involved in the business

Pre-admission consultations

As shown in Table II, the parents of 66 (64.7 percent) of the 102 children had consulted a physician or a nurse prior to admission; however, only 15 of the 66 were referred to OOTH (14 by physicians and one by a nurse) while the remaining 51 as well as 36 others, presented as self-referrals.

Clinical and laboratory features at diagnosis

The frequencies of selected individual clinical features are shown in Table III. Although there were generally no significant differences between patients with BCM and those with BUM, the inci-

dence of bacterial infections localised at other sites approached significance (p = 0.059) (Table III). Thirty six (35.3 percent) of the patients were severely ill on presentation, while 82 (80.4 percent) had received antibiotics and antimalarials either alone or in combination, before presentation. The diagnosis of meningitis was missed on admission in 26 (25.5 percent) of the patients, and only 20.6 percent presented within three days of the onset of their illness.

The aetiological agents and associated illnesses are shown in Table IV. Eighteen (40 percent) of the patients with CM had pneumococcal meningitis, 17 (38 percent) had gram negative bacillary meningitis, and seven (16 percent) others, meningococcal meningitis. The other organisms isolated included gram negative coccobacilli (4 percent) and Staphylococcus aureus (2 percent). A pathogen was isolated by culture in 15 (33.3 percent) of the patients with BCM. The isolates included S. pneumoniae four, N. meningitides one, S. aureus one, Klebsiella spp. one, and untyped coliform spp. eight. The other bacteria which were not grown on culture were identified by Gram staining of the CSF only. As also shown in Table IV, the incidence of associated illnesses was significantly higher in patients with BCM (37.8 percent vs 17.5 percent) than in those with BUM, (p = 0.038). Similarly, the incidence of associated illnesses in patients with coliform meningitis was significantly higher than that in cases with pneumococcal meningitis (p = 0.026) and others with meningococcal meningitis (p

Table III

Clinical Features in 102 Cases of Pyogenic Meningitis

Features	*No (percent) in All Cases (n = 102)*	*No (percent) in Confirmed Meningitis (n = 45)*	*No (percent) in Unconfirmed Meningitis** (n=57)*
Duration of illness			
≤3 days	21 (20.6)	10 (22.2)	11 (19.3)
4-7 days	39 (38.2)	19 (42.2)	20 (35.1)
≥8 days	42 (41.2)	16 (35.6)	26 (45.6)
Diarrhoea	7 (6.9)	4 (8.9)[+]	3 (5.3)
Drugs given			
Antibiotics	15 (14.7)	7 (15.6)	8 (14.0)
Antimalarials	41 (40.2)	19 (42.2)	22 (38.6)
Both	26 (25.5)	10 (22.2)	16 (28.1)
Convulsions	35 (34.3)	17 (37.8)	18 (31.6)
Coma	16 (15.7)	9 (20.0)	7 (12.3)
Focal neurological signs++	8 (7.8)	4 (8.9)	4 (7.0)
Typical meningeal signs absent	22 (21.6)	8 (17.8)	14 (24.6)
Bacterial infections localised at other sites[+]*	16 (15.7)	11 (24.4)	5 (8.8)
Malnutrition[#]			
Undernutrition	39 (38.2)	19 (42.2)	20 (35.1)
Marasmus	7 (6.9)	4 (8.9)	3 (5.3)
Severely ill[##]	36 (35.3)	17 (37.8)	19 (33.3)
Missed diagnosis on admission	26 (25.5)	10 (22.2)	16 (28.1)

* Positive Gram stain/culture of CSF or positive blood culture

** Negative Gram stain/culture of CSF or negative blood culture where this was done

[+] One had dysentery

[++] Hemiparesis, unilateral dilated pupil, etc

[#] Undernutrition: 60-80 percent of expected weight for age, no oedema; marasmus: <60 percent of weight for age, no oedema[1]

[##] Aggregate severity score ≥3/10

[+]* Difference infrequency between confirmed and unconfirmed meningitis approached significance (p = 0.059)

Table IV

Aetiological Agents and Illnesses associated with Pyogenic Meningitis

Aetiologic agents	*No of Cases*	*No (percent) with other Illnesses*	*Associated Illnesses*
GPDC/S. pneum	18	4 (22.2)	Suppurative ottis media 1, infected wound 1, bronchopneumonia 1, diarrhoea 1
GNDC/N. mening	7	1 (14.3)	Marasmus with diarrhoea and Bronchopneumonia
Gram negative coccobacilli	2	1 (50.0)	Pyoarthritis of the knee
Staphylococcus aurues	1	1 (100.0)	Marasmus with osteomyelitis involving multiple sites
Gram negative bacilli	8	3 (37.5)	Infected wound 1, HIV-1 seropositivity 1, diarrhoea 1
Untyped iform spp.	8	6 (75.0)	Dysentery + bronchopneumonia col-1, bronchopneumonia 1, orbital cellulitis 1, marasmus + prolonged obstructive jaundice 1, bronchopneumonia + pyoarthitis 1, marasmus 1
Klebsiella spp	1	1 (100.0)	Recent measles
Unknown*	57	10 (17.5)	Marasmus 3, bronchopneumonia 2, diarrhoea 2, measles with bron-chopneumonia 1, diarrhoea + bronchopneumonia 1, infected wound 1.

GPDC/S. pneum = gram positive aiplococci/S pneumoniae;
GNDC/N. mening = gram negative dip`ɔcocci/N menigitidis
*Negative Gram stain/culture of CSF or negative blood culture where this was done

= 0.041). The numbers of the other agents involved were too small for meaningful analysis.

The incidence of various selected laboratory data is shown in Table V. This shows no significant differences between patients with BCM and those with BUM. Generally, except for malaria parasitaemia, the yield of abnormal test results was low. Only 22.3 percent of the patients had severe meningitis as defined by a CSF glucose of 1.1 mmol/l (20 mg/dl) or less.[8] Tests that did not involve the CSF, except for haematocrit and blood film for malaria parasites, were carried out in less than half of the patients. The severity of parasitaemia was ++ or greater in 33 (68.8 percent) of the patients with malaria parasitaemia.

Table V

Laboratory Data at Diagnosis of Pyogenic Meningitis

Feature	*Number positive/number tested (percent positive)*		
	All Cases	*Confirmed Meningitis*	*Unconfirmed Meningitis***
CSF Gram stain	36/102 (35.3)	36/45 (80)	Not applicable
CSF culture	10/102 (9.8)	10/45 (22.2)	Not applicable
Bacteraemia	5/21 (23.8)	5/10 (50)	0/11
Bacteriuria+	4/18 (22.2)	1/8 (12.5)	3/10 (30.0)
CSF glucose ≤20mg/dl++	23/102 (22.5)	11/45 (24.4)	12/57 (21.1)
Malaria parasitaemia	48/78 (61.5)	22/33 (66.7)	26/45 (57.8)
Haematocrit ≤20%#	13/96 (13.5	7/42 (16.7)	6/54 (11.1)
Peripheral white bloodCount ≥15m000/cu. mm##	11/67 (16.4)	6/30 (20.0)	5/37 (13.5)
Sickle cell disease	1/12 (8.3)	1/10 (10.0)###	0/2
HIV seropositive	1/8 (12.5)	1/6 (16.7)¨	0/2
Widal titre ≥1:160¨¨	3/14 (21.4)	1/3 (33.3)	2/11 (18.2)

* Positive Gram stain/culture of CSF or positive blood culture
** Negative Gram stain/culture of CSF or negative blood culture where this was done
+ All four patients had coliform bacteriuria
-+ Severe meningitis[8]
Severe anaemia[9]
Leucocytosis[10]
Had pneumococcal meningitis
¨ Had gram negative bacillary meningitis
¨¨ Positive Widal test[11]

Outcome of illness

Five patients were discharged against medical advice; one was known to have died about two weeks after discharge. Case fatality rate in the 97 patients with known outcome of hospitalisation was 11.3 percent (11/97). Fifteen (17.4 percent of the survivors or 15.5 percent of the overall cohort of 97) patients had neurological sequelae while 56 (57.7 percent) apparently recovered fully. There was no significant difference in outcome between patients with BCM and those with BUM (case fatality rates were 18.2 percent and 5.7 percent, respectively; relative risk (95 percent confidence interval). RR (95 percent CI) = 3.3 (0.9, 11.6), p = 0.061 while neurological sequelae rates were 19.4 percent and 16.0 percent, respectively [RR (95 percent CI) = 1.2 (0.5, 3.1)], (p = 0.879).

The presenting features with significant effects on outcome are shown in Table VI. They included age, the severity of illness, and the presence of convulsions, coma, focal neurological signs and severe malnutrition. There was no significant relationship between duration of illness and treatment with antibiotics before admission and the outcome. There was also no significant relationship between CSF glucose level, cellular response in the CSF, peripheral leukocytosis, malaria parasitaemia and haematocrit and the outcome. The overall adverse outcome rate was higher in patients with coliform meningitis (7/8 or 87.5 percent) than in those with pneumococcal (3/18 or 16.7 percent; p = 0.001) or meningococcal (2/7 or 28.6 percent; p = 0.41) meningitis. The number of patients with the other aetiological agents was too small for meaningful analysis.

Table VI

Factors influencing Outcome in Meningitis

		percent with adverse outcome			Relative risk (95% confidence interval), P*		
	N	*Death*	*Neurological Sequelae*	*Death or Sequelae*	*Death*	*Sequelae in Survivors*
All patients	97	11.3	15.5	-	-	-
Aggregate severity score: ≥3.0	33	27.3	33.3	6.5(2.9,14.5)	8.7(2,38.1)	7.1(2.5,20.2)
0-2	64	3.1	6.3	<0.0001	<0.001	<0.0001
Age: 1-24 mon	28	17.9	32.1	2.9(1.5,5.4)	2.0(0.7,6.2)	4.1(1.6,10.7)
>24 mon	69	8.7	8.7	0.002	0.287	0.003
1-12 mon	16	25.0	31.3	2.7(1.5,4.9)	2.9(1.0,8.7)	3.1(1.3,7.5)
>12 mon	81	8.6	12.4	0.014	0.08	0.031
Convulsions before diagnosis:						
present	34	23.5	26.5	3.5(1.7,7.0)	4.9(1.4,17.4)	3.5(1.4,8.7)
absent	63	4.8	9.5	0.0004	0.014	0.011
Focal neurologic signs at diagnosis:						
present	7	28.6	57.1	3.9(2.4,6.3)	2.9(0.8,10.7)	5.9(2.9,11.9)
absent	90	10.0	12.2	0.001	0.179	0.003
Coma: present	14	42.9	28.6	3.7(2.1,6.4)	7.1(2.5,20.2)	3.5(1.5,8.6)
absent	83	6.0	13.3	0.0002	0.0001	0.028
Severe malnutrition:						
present	6	33.3	66.7	4.5(3.1,6.7)	3.4(0.9,12.2)	7.4(4.3,12.9)
absent	91	9.9	12.1	0.0002	0.136	0.0006

*X^2 test/Fisher's exact test for risk of stated outcome in those with vs without the stated feature

Discussion

The population in Edo central senatorial zone is probably typical of those in most small urban towns and rural areas in southern Nigeria. Based on the results of the present study, three features appear to be characteristic of PM in children in these areas: a high overall incidence, a lack of decline in incidence with age, and a high incidence of Gram negative bacillary meningitis (GNBM). There is the need to confirm these findings in other areas but for now, two questions arise about PM in rural areas: first, how do these findings compare with what is known about PM in the large urban areas, and, what are the implications of the findings?

Data on the incidence of PM in the large urban towns and cities in Nigeria are available mainly from the reports on the pattern of emergencies. Only a few of the reports dealing specifically with PM give the incidence as a proportion of total admissions, perhaps because many of them dealt with specific aspects of the problem such as aetiological agents, antibiogram, etc. PM has been reported to account for 0.7 percent of emergencies in Calabar,[12] 0.6 percent in Enugu,[13] 2.0 percent in Benin City,[14] 5.1 percent in Ibadan,[15] and 1.4 to 2.4 percent in Lagos.[16] In specific studies on PM, the incidence of admissions due to PM was 3.1 percent in Enugu,[17] 2.8 percent in Sagamu,[18] 6.2 percent in Ibadan,[19] and 3.5 percent in Maiduguri[20] in the meningitis belt; the reports from Sagamu and Ibadan were inclusive of neonates. By

comparison, in Accra, Ghana, one percent of admissions of seriously ill children was due to PM.[21] The incidence of 4.8 percent in the present report is higher than those from these other areas, except Ibadan. The high incidence in Ibadan may be part of the usual intra-regional variation in the incidence of diseases. This may also be a factor in the high incidence observed in the present study. However, we think that other factors, which are presently unknown, may be at play judging by the lack of decline in incidence with age and the high incidence of GNBM.

The lack of decline in incidence with age and the large number of cases involving older children were other peculiar findings in this study. They are in contrast to the findings in the large urban towns and cities in Nigeria[19 20 22-24] and other African countries,[21 25 26] and reports from developed countries.[27] In these areas, PM is predominantly a disease of infants and young children. The present observation may be difficult to explain. As LP rate is higher in young children in our centre (unpublished report), it seems unlikely that a significant number of cases of PM in young children were missed. Furthermore, the possibility that infants and young children may have a more rapidly progressive course ending in death at home is not supported by the high incidence observed in the six months of life. Further studies are required to confirm the pattern of incidence in relation to age and to determine the factors involved as they may have implications for the efforts at prevention.

A high incidence of GNBM was another unusual finding in this study. Compared to developed countries, the incidence of GNBM is high in developing countries.[28] This may be a reflection of the high incidence of Gram negative septicaemia in the tropics[29] in addition to the possible association of GNBM with diarrhoea in developing countries.[25] However, even allowing for these factors, the incidence of 8.8 percent of the total of 102 patients and 20.0 percent of the 45 with BCM in the present series, is quite high, and compares unfavourably with the incidence in large urban areas in developing countries.[28] Further studies are recommended to confirm this observation and to determine possible contributing factors. GNBM is usually associated with a high incidence of adverse outcome[28 30] and the results of the present study confirmed this.

The low case fatality rate of 11.3 percent compares favourably with the 20.7 percent to 24.2 percent reported from large urban centres.[5 18 21 25 31] However, the 17.4 percent sequelae rate in survivors was only slightly lower than the 22 percent to 47.4 percent reported from the large urban areas.[5 17 21-23 31] The factors associated with an adverse outcome in the present series were similar to those reported from the large urban areas[5 25] and from developed countries.[8 27] The principal reason for the lower case fatality rate in this study may therefore, be the age-structure of our patients; mortality is usually higher in infancy,[27] a category of patients that was few in the present series. For the same reason, the sequelae rate was expected to be lower. However, the problems of a high incidence of late presentation, malnutrition, and pneumococcal meningitis and GNBM, all factors associated with an increased incidence of sequelae,[25 27 28 30] may have nullified the favourable effect of age to produce the high sequelae rate.

There was a high rate of pre-admission consultation of physicians and nurses by the parents in this series. However, this appeared not to have had a positive impact in terms of early diagnosis and improved outcome. Many of the patients were commenced on antibiotics without adequate evaluation and this may have been partly responsible for late presentation in many cases. Also, only a few (14.7 percent) were referred to OOTH, although about 65 percent had been seen by a physician or nurse at least once, during the illness before admission. This finding suggests some degree of deficiency in the evaluation and management of patients by physicians in our areas. Although this may be partly due to a heavy work load, it has serious implications. There may be an urgent need for "update courses" on the recognition and management of PM, including the need for early referral of suspected cases directed not only at physicians in general/private practice, but other health workers as well.

Some aspects of the findings in the present series deserve further examination. For example, many patients had co-existing infections such as malaria and other bacterial infections which have the potential to act as red herrings in patients with PM.[32] There were limitations in laboratory diagnosis: blood culture was not done in many of the patients; bacteria grown on culture were not fully typed; there were many instances in which an organism was identified on a Gram stained smear of the CSF but not grown on culture; and it was possible to identify aetiological agents in only about 44 percent of the patients. Treatment with antibiotics before admission and limited ability to pay for investigations may be partly responsible for some of these problems. However, inadequacies in laboratory methods or facilities can not be discounted. These lapses in investigation, limit the amount and quality of available clinical and epidemiological data and are some of the problems in the diagnosis and management of PM in developing countries. Apart from the high incidence of GNBM, the pattern of aetiological agents is similar to that in a recent report from Ghana.[21] The incidence

of clinically obvious neurological sequelae in survivors further confirms the role of PM as an important aetiological factor in severe handicapping conditions in developing countries.[33 34] The incidence of less obvious sequelae in the apparently intact survivors is unknown because of limited follow-up evaluation. Facilities for rehabilitation are inadequate and this underscores the importance of prevention through vaccination, early diagnosis and adequate management.

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